

# Restrictive cardiomyopathy and constrictive pericarditis: non-invasive distinction by digitised M mode echocardiography

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**SUMMARY** It is difficult to distinguish between restrictive cardiomyopathy and constrictive pericarditis on the basis of clinical findings and simple investigation. Cardiac catheterisation has been the reference standard for diagnosis but even this does not always permit an accurate distinction. A Summagraphics digitiser and Prime 750 computer system were used to digitise the echocardiograms of 15 patients with restrictive cardiomyopathy, 10 with constrictive pericarditis and a group of 20 age and sex matched normal subjects of similar age and sex distribution. Compared with controls, patients with restrictive cardiomyopathy showed a significant reduction in the following variables (*a*) decreased fractional shortening, (*b*) decreased peak left ventricular filling and emptying rates, (*c*) decreased percentage posterior wall thickening, and (*d*) decreased peak left ventricular posterior wall thickening and thinning rates. Whereas patients with constrictive pericarditis only had significantly reduced peak left ventricular filling and posterior wall thinning rates and significantly increased posterior wall thinning rate. When patients with restrictive cardiomyopathy were compared with those with constrictive pericarditis the significant differences were: (*a*) decreased peak left ventricular emptying rate, (*b*) decreased percentage posterior wall thickening, and (*c*) decreased peak left ventricular posterior wall thickening and thinning rates.

Digitisation of M mode echocardiograms, with particular attention to posterior wall function, may be a useful adjunct to cardiac catheterisation in distinguishing restrictive cardiomyopathy from constrictive pericarditis.

Impaired ventricular filling may result from atrioventricular valve stenosis or from reduced "ventricular compliance". The latter may itself be due to disease of the pericardium (constrictive pericarditis), disease of the pericardial space (pericardial tamponade), or disease of the myocardium (for example, restrictive or hypertrophic cardiomyopathy). Pericardial tamponade and hypertrophic cardiomyopathy are readily diagnosed by cross sectional echocardiography but pericardial constriction and restrictive cardiomyopathy giving rise to restrictive pathophysiology are not easily distinguished by echocardiography.<sup>1,2</sup> Neither do clinical findings or the results of simple investigation always permit such a distinction. Occasionally, even cardiac catheterisation fails to make the diagnosis.<sup>3</sup>

We describe the features of digitised M mode

echocardiograms that distinguish the restrictive pathophysiology of restrictive cardiomyopathy from that of constrictive pericarditis and which may find clinical application when other diagnostic approaches have failed to distinguish these conditions.

## Patients and methods

Between 1973 and 1987 19 908 patients underwent cardiac catheterisation at the Brompton Hospital. From angiographic and haemodynamic data so obtained restrictive cardiomyopathy was diagnosed in 18 patients (eight women and 10 men, age 23-72, mean 67 years) and constrictive pericarditis in 13 (three women and 10 men, age 19-69, mean 64 years). The criteria used at cardiac catheterisation for diagnosis were as follows:

**Restrictive cardiomyopathy**—Left ventricular angiogram showing predominantly abnormal diastolic function with comparative preservation of systolic function; raised and different left and right

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ventricular end diastolic pressures in the absence of primary valve or congenital heart disease; left ventricular end diastolic pressure usually more raised than right ventricular end diastolic pressure<sup>4,5</sup>;

**Constrictive pericarditis**—Left ventricular angiogram showing predominantly abnormal diastolic function with comparative preservation of systolic function; equal and raised left and right ventricular end diastolic pressures in the absence of primary valve or congenital heart disease; equal diastolic pressures in all four cardiac chambers; and early diastolic dip and plateau pattern (square root sign) in the ventricular pressure trace.<sup>5-7</sup>

The case records of these 31 patients were reviewed and the clinical features and results of simple investigations analysed. The symptoms and clinical signs elicited at presentation together with abnormalities documented on the plain posteroanterior chest radiograph and resting scalar electrocardiogram at presentation were noted.

M mode echocardiograms which had been performed in all patients were also analysed to determine whether the two conditions could be distinguished by non-invasive means. Traces were made with Cambridge Instruments equipment with a 2.25 MHz transducer. All patients had been studied in the left semilateral position with simultaneous electrocardiograms and phonocardiograms recorded at a paper speed of 100 mm/s. Records of the left ventricular cavity used for digitisation were taken at the tips of the mitral valve leaflets. Echocardiograms were considered suitable for digitisation if the M mode recording showed clear leading edge endocardial echoes from the septum and posterior left ventricular wall. This condition was satisfied in 10 patients with constrictive pericarditis and 15 patients with restrictive cardiomyopathy. Echocardiograms were digitised by a Summagraphics digitiser and a Prime 750 computer system. At least three cardiac cycles were analysed for each patient and the mean values calculated. The following variables were measured:

- Left ventricular cavity size was measured both at end diastole (EDD (cm)) and end systole (ESD (cm)) (taken as those dimensions synchronous with the Q wave of the electrocardiogram and A<sub>2</sub> on the phonocardiogram respectively).
- Fractional shortening (FS (%)) was derived:  $FS = (EDD - ESD) \div EDD$ .
- Peak rate of increase of left ventricular dimension during early diastole (LV max rate (cm/s) (this represents peak left ventricular filling rate)).
- Peak rate of reduction of left ventricle dimension during systole (LV min rate (cm/s)) (this represents peak left ventricular emptying rate).
- Posterior wall thickness at minimum cavity size

Table 1 Historical and clinical features

		Constrictive (%)	Restrictive (%)
Duration of history:	> 1 year	10 (83)	4 (22)
	< 1 year	3 (17)	14 (78)
Presenting symptoms:	Chest pain	0 (0)	4 (22)
	Dyspnoea	13 (100)	14 (76)
Findings on physical examination	Raised venous pressure	13 (100)	18 (100)
	Atrial fibrillation	1 (8)	6 (33)
	Systolic murmur	0 (0)	9 (50)
	S3/S4	3 (23)	7 (39)
	Pulmonary venous congestion	0 (0)	9 (50)
Posteroanterior chest radiograph:	Cardiomegaly	2 (15)	8 (44)
	Pericardial calcification	4 (30)	0 (0)
	ST/T wave changes	9 (69)	9 (50)
Scalar electrocardiogram:	Low voltage	5 (38)	0 (0)
	QRS complexes		

(PW min (cm)).

(f) Posterior wall thickness at maximum cavity size (PW max (cm)) and

(g) percentage systolic thickening of posterior wall (%PW) derived from:  $(PW \text{ min} - PW \text{ max}) \div PW \text{ min} \times 100$ .

(h) Peak rate of thinning of posterior wall during early diastole (PW max rate (cm/s)).

(i) Peak rate of thickening of posterior wall during systole (PW min rate (cm/s)).

(j) Septal thickness at minimum cavity size (sept min dim (cm));

Table 2 Haemodynamic data obtained at cardiac catheterisation in patients with restrictive cardiomyopathy

Patient	LV/RV EDP	RA	PW	CI	Biopsy
1	28/25	—	25	—	F
2	20/14	16	20	—	—
3	22/17	17	35	1.4	—
4	28/10	13	36	—	—
5	28/14	30	30	1.5	HF
6	22/—	16	32	—	F
7	30/20	22	30	1.4	—
8	28/19	10	21	—	—
9	44/32	7	27	—	A
10	45/20	15	40	2.2	—
11	40/28	28	35	—	—
12	36/—	22	33	—	A
13	20/14	12	15	—	A
14	22/23	11	8	—	—
15	21/12	8	22	3.1	—
16	35/28	25	31	1.5	—
17	45/18	5	—	—	—
18	30/20	9	—	—	—
Mean	30/20	16	28	1.8	—

LV/RV EDP, left ventricular/right ventricular end diastolic pressure; RA, right atrial pressure; PAW, wedge pressure; CI, cardiac index; H, hypertrophy; F, fibrosis; A, amyloid. All pressures are mm Hg.

Table 3 Haemodynamic data obtained at cardiac catheterisation in patients with constrictive pericarditis

Patient	LV/RV EDP	RA	PW	CI
1	25/25	23	26	2.1
2	13/15	12	12	2.4
3	15/14	14	16	1.9
4	15/15	20	20	1.3
5	17/18	21	25	—
6	23/25	23	23	—
7	20/20	19	—	—
8	17/22	12	22	—
9	19/18	18	19	—
10	28/23	24	23	2.8
11	24/24	20	22	—
12	14/14	12	17	3.6
13	18/18	21	23	—
Mean	18/18	17	18	2.0

LV/RV EDP, left ventricular/right ventricular end diastolic pressure; RA, right atrial pressure; PAW, wedge pressure; CI, cardiac index. All pressures are mm Hg.

(k) septal thickness at maximum cavity size (sept max dim (cm)).

These variables were also measured in a control group of 20 patients of similar age range and sex distribution who had no clinical cardiac disease and a negative stress test at high workload.

### Statistical methods

Data are expressed as mean (1 SD). Mean values were compared by Student's *t* test and the relative importance of discriminating variables determined by logistic regression.

### Results

Table 1 summarises the clinical features of both groups. Dyspnoea was the major presenting symptom in both groups but the history of this was often shorter in the group with restrictive cardiomyopathy. Physical examination showed raised venous pressure in all patients. Atrial fibrillation occurred in 33% of patients with restrictive cardiomyopathy but in only

8% of patients with constrictive pericarditis. A pansystolic murmur was heard only in patients with restrictive cardiomyopathy but an added diastolic noise was equally common in both groups.

Radiographic evidence of upper lobe blood diversion and left atrial enlargement on the postero-anterior chest radiograph were seen only in patients with restrictive cardiomyopathy; calcification on the lateral chest radiograph seemed to be specific for constrictive pericarditis but occurred in only 30% of such patients. Repolarisation (ST/T wave) changes occurred more commonly in patients with constrictive pericarditis (69% compared with 50% of patients with restrictive cardiomyopathy) while low voltage QRS was found in this group alone but in only 38% of patients.

Tables 2 and 3 show the results at cardiac catheterisation. These data were used to determine diagnosis according to the criteria listed above (see Methods). Those patients with restrictive cardiomyopathy had raised end diastolic pressures in both the right and left ventricles but with a greater increase in the left ventricular end diastolic pressure (left ventricular end diastolic pressure mean 30, range 28–45 mm Hg; right ventricular end diastolic pressure mean 20, range 12–32 mm Hg). Patients with constrictive pericarditis had raised and equal diastolic pressures in all four cardiac chambers. In our series, mean ventricular end diastolic pressure was lower in patients with constrictive pericarditis (mean left ventricular end diastolic pressure 18 mm Hg, mean right ventricular end diastolic pressure 18 mm Hg) than in patients with restrictive cardiomyopathy (mean left ventricular end diastolic pressure 30 mm Hg, mean right ventricular end diastolic pressure 20 mm Hg).

Left ventricular biopsy was performed in 30% of patients with restrictive cardiomyopathy; this confirmed the aetiology in half of them.

### DIGITISED M MODE ECHOCARDIOGRAPHY (TABLE 4)

Figure 1 shows typical examples of digitised traces

Table 4 Digitisation of M mode echocardiograms

Variable	Restrictive	<i>p</i>	Constrictive	<i>p</i>	Controls
LV% change	22 (9.8)	< 0.001*	30.5 (10.2)		36 (5.8)
LVmax rate (cm/s)	7.5 (3.3)	< 0.001*	8.8 (2.5)	< 0.004*	12 (2.6)
LVmin rate (cm/s)	-5.3 (1.7)	< 0.001*	-8.5 (2.8)		-8.3 (1.1)
PW%	24% (12)	< 0.006†	47% (15)		35% (7)
PWmax rate (cm/s)	2.6 (0.9)	< 0.007*	6 (2.8)	< 0.01*	3.4 (0.7)
PWmin rate (cm/s)	-4.1 (2.5)	< 0.008†	-6.7 (1.9)		-5.8 (1.8)

\*Test of significance against controls.

†Test of significance against group with constrictive pericarditis.

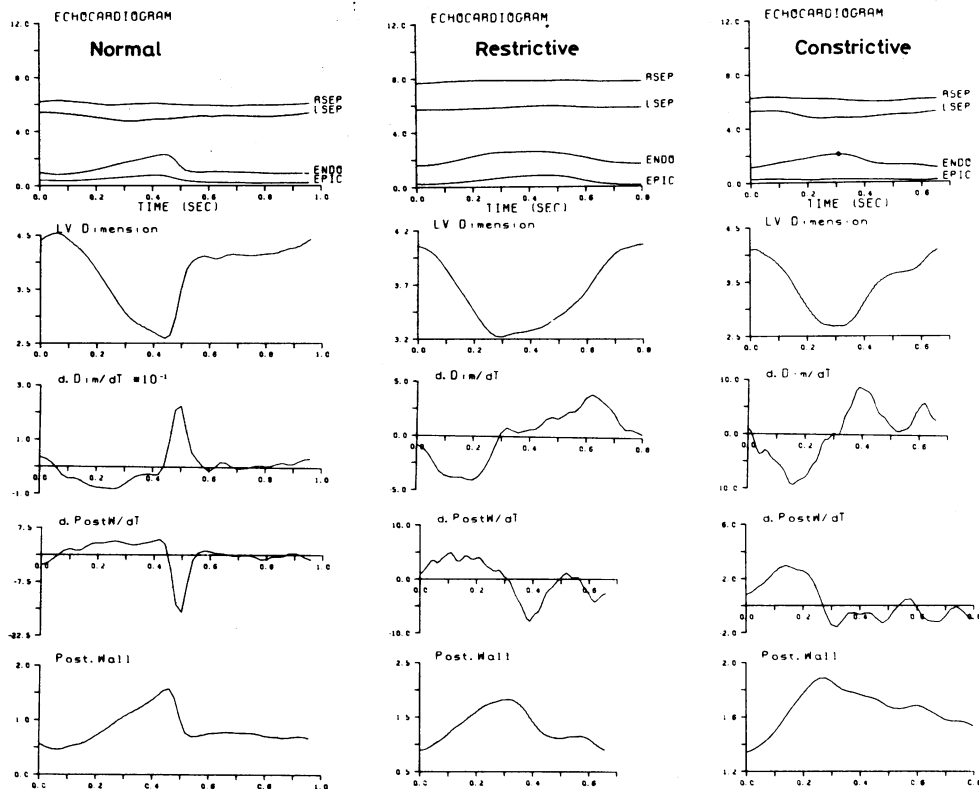


Fig 1 Typical examples of computer printouts of digitised echocardiograms from a control, from a patient with restrictive cardiomyopathy, and from a patient with constrictive pericarditis. R, right; L, left; SEP, septum; ENDO, endocardium; EPIC, epicardium; LV, left ventricle;  $d \text{ Dim}/dT \times 10^{-1}$ , rate of change in left ventricular dimension;  $d \text{ PostW}/dT$ , rate of change in thickness of posterior wall.

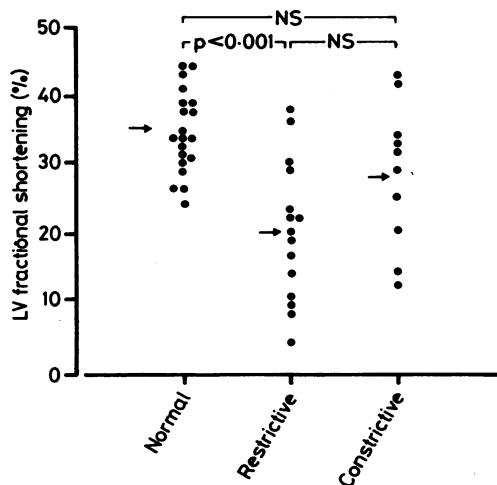


Fig 2 Left ventricular fractional shortening (LV%) (see text) in controls, patients with restrictive cardiomyopathy, and patients with constrictive pericarditis.

obtained from controls, patients with restrictive cardiomyopathy, and patients with constrictive pericarditis.

There was no significant change in left ventricular cavity dimension at end systole or diastole in either restrictive cardiomyopathy or constrictive pericarditis as compared with control patients.

Fractional shortening was significantly less in the group with restrictive cardiomyopathy than in the controls (mean  $22 (9.8) \% v 36 (5.8) \%$ ,  $p < 0.001$ ) but compared with the group with constrictive disease the difference did not achieve statistical significance (mean  $31 (10.2) \% v 36 (5.8) \%$ ). There was no significant difference in fractional shortening between the groups with restrictive cardiomyopathy and constrictive pericarditis (fig 2). Peak rate of increase of cavity dimension in diastole (LV max rate, that is, peak ventricular filling rate) was significantly lower in both constrictive pericarditis (mean  $8.8 (2.5) \text{ cm/s}$ ,  $p < 0.003$ ) and restrictive cardiomyopathy (mean  $7.5 (3.3) \text{ cm/s}$ ,  $p < 0.001$ )

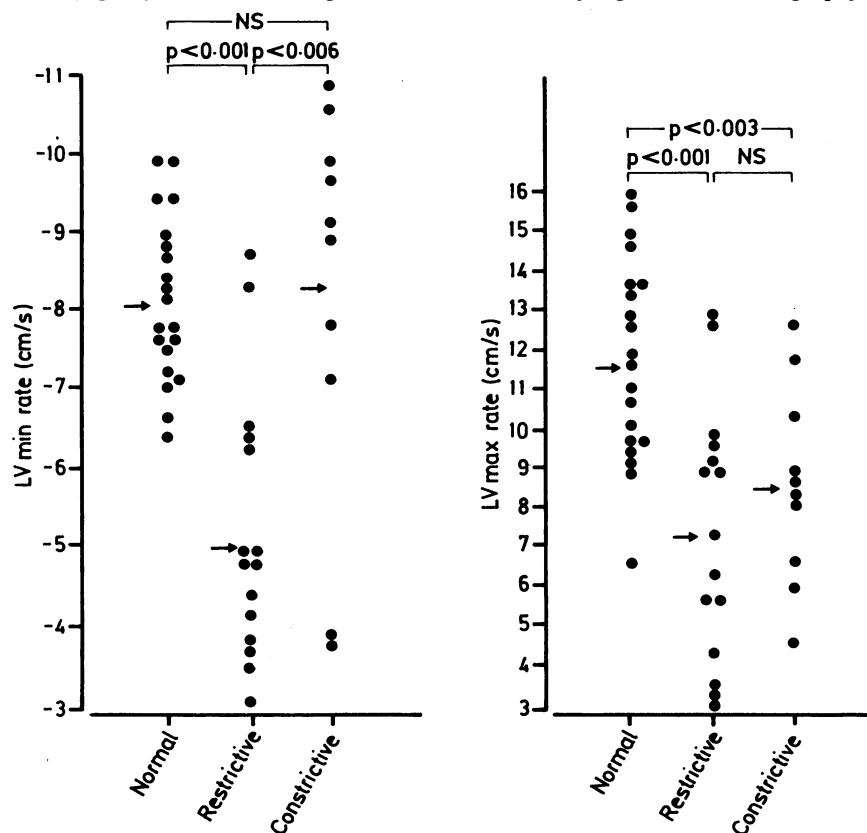


Fig 3 Peak rate of reduction of left ventricular dimension during systole (LV min rate (cm/s)) and peak rate of change of left ventricular dimension during early diastole (LV max rate (cm/s)) (see text) in controls, patients with restrictive cardiomyopathy, and patients with constrictive pericarditis.

than in the controls (fig 3) but this variable was not significantly different in the restrictive cardiomyopathy group and the constrictive pericarditis group. The peak rate of decrease of left ventricular dimension during systole (LV min rate)—that is, peak ventricular emptying rate) for the restrictive cardiomyopathy group (mean  $-5.31$  ( $1.7$ ) cm/s) was significantly lower than in the controls (mean  $-8.3$  ( $1.1$ ) cm/s,  $p < 0.001$ ) and those with constrictive pericarditis (mean  $-8.5$  ( $2.8$ ) cm/s,  $p < 0.006$ ) but the difference between those with constrictive pericarditis and controls was not significant (fig 3).

Posterior wall thickness at minimum cavity size (PW min) and at maximum cavity size (PW max) was not significantly different between the various groups but the percentage posterior wall thickening (% PW) was lower in the group with restrictive cardiomyopathy (mean  $24$  ( $12\%$ )) than in either the controls (mean  $35$  ( $7\%$ ),  $p < 0.007$ ) or the group with

constrictive pericarditis (mean  $47$  ( $15\%$ ),  $p < 0.008$ ); again the group with constrictive pericarditis did not differ significantly from the controls (fig 4). The peak rate of thinning of the posterior wall during early diastole (PW max rate) was lower in the group with restrictive cardiomyopathy (mean  $2.6$  ( $0.9$ ) cm/s) than in either the controls (mean  $3.4$  ( $0.7$ ) cm/s,  $p < 0.008$ ) or the group with constrictive pericarditis ( $6$  ( $2.8$ ) cm/s,  $p < 0.004$ ), in which the rate was significantly faster than in controls ( $p < 0.01$ ) (fig 5). Peak rate of thickening of the posterior wall during systole (PW min rate) cm/s was also lower in the group with restrictive cardiomyopathy (mean  $-4.1$  ( $2.5$ ) cm/s) than in either the controls ( $-5.8$  ( $1.8$ ) cm/s,  $p < 0.02$ ) or the group with constrictive pericarditis ( $-6.7$  ( $1.9$ ) cm/s,  $p < 0.007$ ) but those with constrictive pericarditis were not significantly different from normal (fig 5). It was of interest that the three patients with amyloid disease in the group

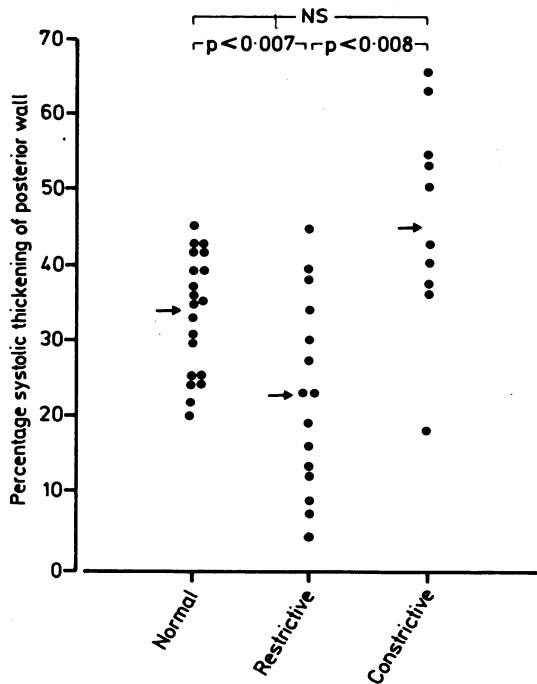


Fig 4 Percentage systolic thickening of posterior wall (see text) in controls, patients with restrictive cardiomyopathy, and patients with constrictive pericarditis.

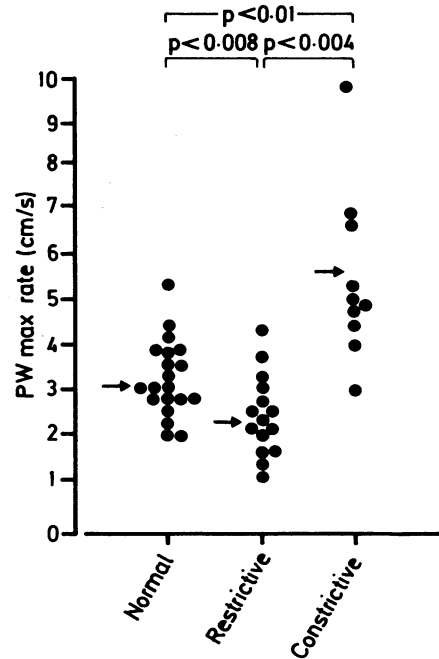
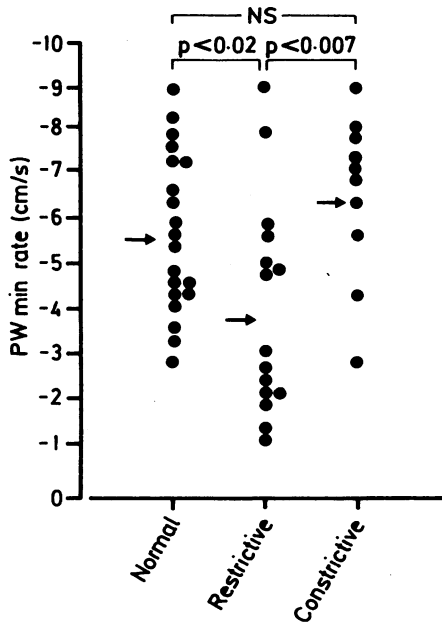


Fig 5 Peak rate of thinning of posterior wall during early diastole (PW max rate) and peak rate of thickening of posterior wall during systole (PW min rate) (see text) in controls, patients with restrictive cardiomyopathy, and patients with constrictive pericarditis.

with restrictive cardiomyopathy were among those patients with the most profound abnormalities of posterior wall variables.

Digitisation of the septum is technically difficult because the leading edge endocardial echoes on the right side of the ventricular septum may be poorly defined. In eight patients, however, this measurement was made and septal dimension during diastole (sept min) was significantly greater in the group with restrictive cardiomyopathy (mean 1.3 (0.5) cm) than in patients with constrictive pericarditis (mean 0.9 (0.4) cm,  $p < 0.01$ ) or controls (mean 0.9 (0.2) cm,  $p < 0.01$ ). However, the maximum septal dimension in both disease groups was not significantly different from control values.

We applied a logistic regression analysis to those variables that were significantly different in restrictive cardiomyopathy and constrictive pericarditis. This showed that peak rate of thinning of the posterior wall during early diastole was the best discriminant. No other variable made a significant contribution once this variable was included. This was because peak rate of thinning of the posterior wall during early diastole was significantly associated with all the other independent variables, in particular peak rate of decrease of left ventricular dimension during early systole, percentage posterior wall thickening, and peak rate of thickening of posterior wall during early systole ( $p < 0.001$  for each correlation).

## Discussion

Impairment of ventricular diastolic function with comparative preservation of systolic function is characteristic of restrictive cardiomyopathy<sup>8</sup> and it is this pre-eminence of diastolic dysfunction that distinguishes this condition from other myocardial disorders in which impaired systolic function is the major abnormality. Constriction of the pericardium, however, also primarily impairs ventricular diastolic function rather than systolic function<sup>3,9,10</sup> and hence distinguishing between these two conditions is important, particularly since restrictive cardiomyopathy can only be treated symptomatically whereas the symptoms and signs of pericardial constriction can be dramatically alleviated by pericardectomy.<sup>11</sup>

Because they have a similar pathophysiology it is not surprising that historical features and findings on physical examination<sup>9-11</sup> do not necessarily permit a clinical distinction. In our series most patients with either restrictive cardiomyopathy or constrictive pericarditis presented with a history of progressive dyspnoea, though those with restrictive cardiomyopathy tended to have had symptoms for a shorter time. Physical signs were also similar in both groups—venous pressure was raised in all patients but atrial fibrillation occurred in only 8% of patients with constrictive pericarditis and 33% of patients with restrictive cardiomyopathy. The murmurs of mitral or tricuspid valve incompetence have been widely reported in restrictive cardiomyopathy,<sup>10,11</sup> whereas constrictive pericarditis is characteristically associated with a “quiet” heart.<sup>3</sup> There are, however, sporadic reports of murmurs caused by atrio-ventricular valve incompetence in constrictive pericarditis.<sup>12</sup> In our series pansystolic murmurs were heard only in patients with restrictive cardiomyopathy. An early diastolic sound may occur in either condition though some would attribute a specific quality to this sound in constrictive pericarditis.<sup>13</sup> This sound is thought to be caused by sudden deceleration in ventricular filling as a consequence of external restriction. In our series this physical sign was seen in 23% of patients with constrictive pericarditis and 39% of patients with restrictive cardiomyopathy.

Simple investigations may help to establish a diagnosis in some cases. Calcification in the pericardium, best seen on the lateral chest radiograph, is highly specific for constrictive pericarditis but is frequently absent.<sup>9</sup> We found pericardial calcification in only 27% of patients with constrictive pericarditis and it was never recorded in patients with restrictive cardiomyopathy. Though repolarisation abnormalities (ST/T wave changes) have also

been reported in constrictive pericarditis they are considered to be non-specific and insensitive, and this was our experience.<sup>9</sup> Though we found low voltage QRS complexes in only the group with constrictive pericarditis they were present in only 33% of patients. It is clear then that clinical observations and simple investigations do not always allow these two conditions to be distinguished.

In our series cardiac catheterisation and echocardiography permitted a diagnosis to be made. At cardiac catheterisation features of the diastolic pressure trace and diastolic pressure measurements have been specifically associated with restrictive cardiomyopathy<sup>4,5</sup> or constrictive pericarditis<sup>6,7</sup> (as noted in the methods section). The “dip and plateau” waveform of the diastolic ventricular pressure trace is said to be characteristic of constrictive pericarditis, but not surprisingly it may also occur in restrictive cardiomyopathy since both diseases share a common pathophysiology.<sup>10,14,15</sup> In certain cases of constrictive pericarditis manoeuvres such as rapid volume infusion at catheterisation may bring out a dip and plateau waveform which is otherwise inapparent.<sup>16</sup> Diastolic equalisation of pressures throughout the cardiac chambers is regarded as a characteristic of constrictive pericarditis but it can also occur in restrictive cardiomyopathy though a difference between left and right ventricular end diastolic pressures; it is more common for left ventricular end diastolic pressures to be higher than right pressure.<sup>17</sup> In one patient in our series (patient 11, table 2) constrictive pericarditis was diagnosed at first cardiac catheterisation and he underwent pericardial resection. It later became apparent that there had been no clinical improvement, and after repeat echocardiography and cardiac catheterisation with endomyocardial biopsy the diagnosis was revised to restrictive cardiomyopathy. Thus if diagnostic uncertainty persists after cardiac catheterisation there may be no alternative but to consider explorative thoractomy<sup>3</sup>—haemodynamic data do not always lead to diagnosis.

The sensitivity and specificity of endomyocardial biopsy in these groups are undecided.<sup>18,19</sup> While biopsy was performed in 33% of our patients with restrictive cardiomyopathy it was not performed in any of the patients with the diagnosis of constrictive cardiomyopathy and so we are unable to comment on the value of histological features that may serve to distinguish between the two. It is noteworthy, however, that in one of our cases the diagnosis of amyloidosis rested on the observation of a single island of amyloid deposit in a single section and that it is known that the diffuse nature of amyloidosis makes biopsy diagnosis unreliable.

The M mode and cross sectional echocardi-

graphic features of both conditions have been reported but there is no agreement on the features that distinguish the two groups.<sup>2,20,21</sup> Pericardial thickness has been considered as a marker for constrictive pericarditis<sup>22</sup> but it correlates poorly with the findings at operation, and was not a variable that we have found useful.<sup>23</sup> Janos and colleagues suggested the value of computerised digitisation of M mode echocardiograms in distinguishing between constrictive pericarditis and restrictive cardiomyopathy, but they studied only seven patients.<sup>24</sup> Digitised echocardiography has also been used to characterise abnormalities in cardiac amyloidosis.<sup>25</sup> Cross sectional echocardiographic features of amyloidosis have been the subject of many reports: myocardial echo intensity and increased thickness of the atrial walls may be moderately sensitive and highly specific.<sup>26</sup> Though an increase in myocardial echo intensity is a feature in our patients with amyloidosis there are only three such patients and abnormality of echo amplitude is not a feature in others with restrictive cardiomyopathy. We found no other features on cross sectional echocardiography that enabled us reliably to distinguish the two groups from one another or from the controls.

We showed that there are features on digitisation of M mode echocardiograms that distinguish restrictive cardiomyopathy from constrictive pericarditis and from normal patients. Thus fractional shortening, peak rate of increase of left ventricular dimension during diastole, peak rate of reduction of left ventricular dimension during systole, percentage change in posterior wall dimension, and rate of posterior wall thinning and thickening are all highly significantly lower in the group with restrictive cardiomyopathy group than in the controls. These changes indicate that the group with restrictive cardiomyopathy had slower ventricular filling and emptying (with an associated reduced amplitude of wall motion and rate of posterior wall thinning and thickening) than the controls.

Peak rate of reduction of left ventricular dimension during systole was significantly lower in patients with restrictive cardiomyopathy than in controls—that is ventricular contraction in restrictive cardiomyopathy has a slower peak rate. Also the percentage change of posterior wall dimension is significantly reduced so that in restrictive cardiomyopathy the posterior wall thins and thickens less than in constrictive pericarditis. The peak rate of posterior wall thinning and thickening is significantly slower in restrictive cardiomyopathy than in constrictive pericarditis and indeed posterior wall thinning was significantly faster in the patients with constrictive pericarditis than in the controls. These differences give insight into the abnormalities of left ventricular

function, both systolic and diastolic, that occur in these disease processes. In restrictive cardiomyopathy the myocardium is abnormal with reduced compliance. In constrictive pericarditis myocardium and myocardial compliance are normal, permitting rapid early diastolic filling but this is abruptly terminated by the limit imposed by the constricting pericardium. Digitisation of the M mode echocardiogram may also find a clinical application when other methods have failed to distinguish these two conditions. Analysis by logistic regression suggests that for diagnostic purposes peak rate of thinning of the posterior wall during early diastole is the best discriminant. Distinction between restrictive cardiomyopathy and constrictive pericarditis will not be enhanced by the inclusion of other variables because the peak rate of thinning of the posterior wall during early diastole is significantly associated with all other independent variables and in particular with peak rate of decrease of left ventricular dimension during early systole, percentage posterior wall thickening, and peak rate of thickening of posterior wall during early systole.

The history, physical examination, and simple investigations cannot be used to distinguish between restrictive cardiomyopathy and constrictive pericarditis. A diagnosis can usually be made at cardiac catheterisation by careful pressure recording from the four cardiac chambers (indirectly by means of the pulmonary wedge pressure from the left atrium) but unfortunately there can still be overlaps. If the diagnosis is in doubt, digitisation of M mode echocardiograms, with particular attention to posterior wall function, may improve the distinction between these conditions.

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